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NEWS	15	MAR 31	CAS REGISTRY enhanced with additional experimental spectra
NEWS	16	MAR 31	CA/CAPLUS and CASREACT patent number format for U.S. applications updated
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NEWS	20	APR 15	WPIDS, WPINDEX, and WPIX enhanced with new predefined hit display formats
NEWS	21	APR 28	EMBASE Controlled Term thesaurus enhanced
NEWS	22	APR 28	IMSRSEARCH reloaded with enhancements
NEWS EXPRESS	FEBRUARY 08 CURRENT WINDOWS VERSION IS V8.3, AND CURRENT DISCOVER FILE IS DATED 20 FEBRUARY 2008		
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=> s (S6 kinase 1) (3A) (rapamycin)

L1 78 (S6 KINASE 1) (3A) (RAPAMYCIN)

=> s (S6 kinase 2) (3A) (rapamycin)

L2 1 (S6 KINASE 2) (3A) (RAPAMYCIN)

=> d l2 bib ab

L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2005:1337258 CAPLUS

DN 144:106331

TI S6 kinase 2 potentiates interleukin-3-driven cell proliferation

AU Cruz, Rebecca; Hedden, Lee; Boyer, Derek; Kharas, Michael G.; Fruman, David A.; Lee-Fruman, Kay K.

CS Department of Biological Sciences, California State University at Long Beach, Irvine, USA

SO Journal of Leukocyte Biology (2005), 78(6), 1378-1385

CODEN: JLBIE7; ISSN: 0741-5400

PB Federation of American Societies for Experimental Biology

DT Journal

LA English

AB Interleukin-3 (IL-3) mediates hematopoietic cell survival and proliferation via several signaling pathways such as the Janus kinase/signal transducer and activator of transcription pathway, mitogen-activated protein kinase (MAPK) pathway, and phosphoinositide-3 kinase (PI-3K) pathway. Mammalian target of rapamycin (mTOR) is one of the downstream targets of the PI-3K pathway, and it plays an important role in hematopoiesis and immune cell function. To better elucidate how mTOR mediates proliferation signals from IL-3, the authors assessed the role of S6 kinase 2 (S6K2), one of the downstream targets of mTOR, in IL-3 signaling. The authors show that S6K2 is activated by IL-3 in the IL-3-dependent Ba/F3 cell line and that this is mediated by mTOR and its upstream activator PI-3K but not by the MAPK kinase/extracellular signal-regulated kinase pathway. S6K2 is also activated in primary mouse bone marrow-derived mast cells upon IL-3 stimulation. Expression of a rapamycin-resistant form of S6K2, T388E, in Ba/F3 cells provides a proliferation advantage in the absence or presence of rapamycin,

indicating that S6K2 can potentiate IL-3-mediated mitogenic signals. In cells expressing T388E, rapamycin still reduces proliferation at all doses of rapamycin, showing that mTOR targets other than S6K2 play an important role in IL-3-dependent proliferation. Cell-cycle anal. shows that T388E-expressing Ba/F3 cells enter S phase earlier than the control cells, indicating that the proliferation advantage may be mediated by a shortened G1 phase. This is the first indication that S6K2 plays a role in IL-3-dependent cell proliferation.

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